A. Harris
521742

```
=> fil reg
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                      ENTRY      SESSION
FULL ESTIMATED COST                                    0.15         0.15

FILE 'REGISTRY' ENTERED AT 14:41:02 ON 17 APR 2001
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  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

Structure search limits have been increased.  See HELP SLIMIT
for details.

=> s (enamelin or amelogenin or amelin or tuftelin)/cn
             0 ENAMELIN/CN
             0 AMELOGENIN/CN
             0 AMELIN/CN
             0 TUFTELIN/CN
L1           0 (ENAMELIN OR AMELOGENIN OR AMELIN OR TUFTELIN)/CN

=> fil medl,caplus,biosis,embase,wpids,scisearch,ntis
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                      ENTRY      SESSION
FULL ESTIMATED COST                                   15.51        15.66

FILE 'MEDLINE' ENTERED AT 14:41:36 ON 17 APR 2001

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Prepared by M. Hale 308-4258

```
=> s (enamelin? or enamel? or amelogenin? or amelin? or tuftelin?) and
(apoptos? or capase or cell death or clonal delet?)
L2          37 FILE MEDLINE
L3          30 FILE CAPLUS
L4          35 FILE BIOSIS
L5          33 FILE EMBASE
L6           1 FILE WPIDS
L7          39 FILE SCISEARCH
L8           0 FILE NTIS

TOTAL FOR ALL FILES
L9         175 (ENAMELIN? OR ENAMEL? OR AMELOGENIN? OR AMELIN? OR TUFTELIN?)
               AND (APOPTOS? OR CAPASE OR CELL DEATH OR CLONAL DELET?)

=> s l9 and (neoplasm? or cancer or tumour or tumor)
L10          7 FILE MEDLINE
L11          7 FILE CAPLUS
L12          3 FILE BIOSIS
L13          2 FILE EMBASE
L14          1 FILE WPIDS
L15          3 FILE SCISEARCH
L16          0 FILE NTIS

TOTAL FOR ALL FILES
L17         23 L9 AND (NEOPLASM? OR CANCER OR TUMOUR OR TUMOR)

=> dup rem l17
PROCESSING COMPLETED FOR L17
L18          11 DUP REM L17 (12 DUPLICATES REMOVED)

=> d cbib abs 1-11;s hammarstrom l?/au,in;s lyngstadaas s?/au,in;s gestrelius
s?/au,in

L18  ANSWER 1 OF 11  CAPLUS  COPYRIGHT 2001 ACS
2001:239233  Vitamin D and genomic stability.  Chatterjee, M. (P.O. Box 17082,
     Division of Biochemistry, Department of Pharmaceutical Technology,
     Jadavpur University, 700032, Calcutta, India).  Mutat. Res., 475(1-2),
     69-87 (English) 2001.  CODEN: MUREAV.  ISSN: 0027-5107.  Publisher:
     Elsevier Science B.V..
AB   1.alpha.,25-dihydroxyvitamin D3 [1,25(OH)2D3] has been shown to act on
     novel target tissues not related to calcium homeostasis.  There have been
     reports characterizing 1,25(OH)2D3 receptors and activities in diverse
     tissues such as brain, pancreas, pituitary, skin, muscle, placenta,
immune
     cells and parathyroid.  The receptor hormone complex becomes localized in
     the nucleus, and undergoes phosphorylation by reacting with a kinase.
     This form of the receptor then interacts with the Vitamin D responsive
     element of target gene and modifies the transcription of those genes to
     develop the action.  The modulation of gene transcription results in
     either the induction or repression of specific mRNAs (m-RNAs), ultimately
     resulting in changes in protein expression needed to produce biol.
     responses.  Genes for carbonic anhydrase that are expressed at high
levels
```

in osteoclast are known to be involved in bone resorption and Id genes role in osteoblast-osteoclast differentiation reflects the genomic effect of Vitamin D on bones. Genomic action of Vitamin D also explains the biosynthesis of oncogenes, polyamines, lymphokines and calcium binding proteins. However, there is a possibility that some of the actions of 1,25(OH)2D3 may be mediated by non-genomic mechanisms and may not require the binding to Vitamin D receptor (VDR).Vitamin D offers a protection from genotoxic effects of Vitamin D deficiency by increasing the insulin receptor gene expression and BSP (bone sialoprotein), bone-remodeling by decreasing the osteopontin (OPN) m-RNAs, maintaining the normal epidermal structure and **enamel** matrix. Gonadal insufficiency in Vitamin D deficiency was cor. by vitamin mediated direct regulation of the expression of aramotase gene. The supportive role of Vitamin D in placental function is also evident by its influence on human placental lactogen (hpl) gene transcription accompanied by increase hpl m-RNA levels. Further role of Vitamin D is envisaged in identifying cyclin C as an important target for Vitamin D in cell-cycle regulation.Vitamin D at physiol. concn. has been found to protect cell proteins and membranes against oxidative stress by inhibiting the peroxidative attack on membrane lipids. Vitamin D, at a concn. range of 2.times.10-8-5.times.10-8 M, induces **apoptosis** in most **cancer** cells, stabilizes chromosomal structure and prevents DNA double-strand breaks induced either by endogenous or exogenous factors. Vitamin D is also effective in stimulating DNA synthesis in adult alveolar II cells and provides a novel mechanism of modulation of epithelial cell proliferation in the context of lung development and repair against injury. The regulation of various proto-oncogenes (c-myc, c-fos, c-jun), differentiation inducing properties, antiproliferative effects on keratinocytes and inhibitory effects in several human malignancy ranks Vitamin D as a novel hormone that may have physiol. and clin. implication in the carcinogenic process.

L18 ANSWER 2 OF 11 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 1

2000:645865 Document No. 133:242570 **Enamel** matrix protein compositions for antitumor induction of **apoptosis**. Lyngstadaas, Stale Petter; Hammarstrom, Lars; Gestrelius, Stina (Biora Bioex Ab, Swed.). PCT Int. Appl. WO 2000053196 A1 20000914, 36 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-IB245 20000309. PRIORITY: DK 1999-336 19990310.

AB **Enamel** matrix, **enamel** matrix derivs. and/or **enamel** matrix proteins or peptides may be used as therapeutic or prophylactic agents for inducing programmed **cell death** (**apoptosis**), in particular in the treatment or prevention of **cancer** or malignant or benign **neoplasms**.

L18 ANSWER 3 OF 11 MEDLINE
2001067434 Document Number: 20480560. Edar/Eda interactions regulate **enamel** knot formation in tooth morphogenesis. Tucker A S; Headon D J; Schneider P; Ferguson B M; Overbeek P; Tschopp J; Sharpe P T. (MRC Centre for Developmental Neurobiology, King's College, Guy's Hospital, London Bridge, London SE1 1UL, UK.) DEVELOPMENT, (2000 Nov) 127 (21) 4691-700. Journal code: ECW. ISSN: 0950-1991. Pub. country: ENGLAND: United Kingdom. Language: English.

AB tabby and downless mutant mice have apparently identical defects in teeth, hair and sweat glands. Recently, genes responsible for these spontaneous mutations have been identified. downless (Dl) encodes Edar, a novel member of the **tumour** necrosis factor (TNF) receptor family, containing the characteristic extracellular cysteine rich fold, a single transmembrane region and a death homology domain close to the C terminus. tabby (Ta) encodes ectodysplasin-A (Eda) a type II membrane protein of the TNF ligand family containing an internal collagen-like domain. As predicted by the similarity in adult mutant phenotype and the structure of the proteins, we demonstrate that Eda and Edar specifically interact in vitro. We have compared the expression pattern of Dl and Ta in mouse development, taking the tooth as our model system, and find that they are not expressed in adjacent cells as would have been expected. Teeth develop by a well recorded series of epithelial-mesenchymal interactions, similar to those in hair follicle and sweat gland development, the structures found to be defective in tabby and downless mice. We have analysed the downless mutant teeth in detail, and have traced the defect in cusp morphology back to initial defects in the structure of the tooth **enamel** knot at E13. Significantly, the defect is distinct from that of the tabby mutant. In the tabby mutant, there is a recognisable but small **enamel** knot, whereas in the downless mutant the knot is absent, but **enamel** knot cells are organised into a different shape, the **enamel** rope, showing altered expression of signalling factors (Shh, Fgf4, Bmp4 and Wnt10b). By adding a soluble form of Edar to tooth germs, we were able to mimic the tabby **enamel** knot phenotype, demonstrating the involvement of endogenous Eda in tooth development. We could not, however, reproduce the downless phenotype, suggesting the existence of yet another ligand or receptor, or of ligand-independent activation mechanisms for Edar. Changes in the structure of the **enamel** knot signalling centre in downless tooth germs provide functional data directly linking the **enamel** knot with tooth cusp morphogenesis. We also show that the Lef1 pathway, thought to be involved in these mutants, functions independently in a parallel pathway.

L18 ANSWER 4 OF 11 MEDLINE DUPLICATE 2
2000145959 Document Number: 20145959. The cadherin-catenin complex is expressed alternately with the adenomatous polyposis coli protein during rat incisor amelogenesis. Sorkin B C; Wang M Y; Dobeck J M; Albergo K L;

Skobe Z. (The Forsyth Institute, Boston, Massachusetts, USA.) JOURNAL OF HISTOCHEMISTRY AND CYTOCHEMISTRY, (2000 Mar) 48 (3) 397-406. Journal code: IDZ. ISSN: 0022-1554. Pub. country: United States. Language: English.

AB E-cadherin, a calcium-dependent cell-cell adhesion molecule, is expressed in highly specific spatiotemporal patterns throughout metazoan development, notably at sites of embryonic induction. E-cadherin also plays a critical role in regulating cell motility/adhesion, cell proliferation, and **apoptosis**. We have used the continuously erupting rat incisor as a system for examining the expression of E-cadherin and the associated catenins [alpha-, beta-, gamma-catenin (plakoglobin) and p120(ctn)] during amelogenesis. Using immunhistochemical techniques, we observed expression of alpha-catenin and gamma-catenin in ameloblasts throughout amelogenesis. In contrast, expression of E-cadherin, beta-catenin, and p120(ctn) was strong in presecretory, transitional, and reduced stage ameloblasts (Stages I, III, and V) but was dramatically lower in secretory and maturation stage ameloblasts (Stages II and IV). This expression alternates with the expression pattern we previously reported for the adenomatous polyposis coli protein (APC), a **tumor** suppressor that competes with E-cadherin for binding to beta-catenin. We suggest that alternate expression of APC and the cadherin-catenin complex is critical for the alterations in cell-cell adhesion and other differentiated cellular characteristics, such as cytoskeletal alterations, that are required for the formation of **enamel** by ameloblasts.

L18 ANSWER 5 OF 11 MEDLINE

2000450173 Document Number: 20458167. Cytostatic action of **enamel** matrix derivative (EMDOGAIN) on human oral squamous cell carcinoma-derived SCC25 epithelial cells. Kawase T; Okuda K; Yoshie H; Burns D M. (Department of Pharmacology, Faculty of Dentistry, Niigata University, Japan.) JOURNAL OF PERIODONTAL RESEARCH, (2000 Oct) 35 (5) 291-300. Journal code: JMQ. ISSN: 0022-3484. Pub. country: Denmark. Language: English.

AB During surgical treatment of periodontal disease, **enamel** matrix derivative (EMD) is topically applied as a substitute for extracellular matrix in order to facilitate regeneration of damaged periodontal tissue. However, the mechanism for EMD action is poorly understood. We have now examined the effects of EMD on the proliferation of oral epithelial (SCC25) cells in vitro. After 3 days of treatments, EMD (25 100 microg/ml) dose-dependently inhibited cell division and concomitantly arrested cell cycle at the G1 phase. Prior to this inhibition, EMD significantly up-regulated p21WAF1/cip1, a cyclin-dependent kinase inhibitor, induced G1-arrest, and inhibited DNA synthesis. In addition, EMD down-regulated expression of cytokeratin-18 (CK18) protein, which was most due to decreased production, but less to increased degradation. However, EMD did not discernibly increase the number of apoptotic cells over 8 days of treatment. These findings indicate (1) that EMD acts as a cytostatic agent, rather than a cytotoxic agent, on epithelial cells, and (2) that this anti-proliferative action is probably due to p21WAF1/cip1-mediated G1-arrest. Furthermore, our in vitro cellular data clearly verify and

provide an explanation for the clinical observation that EMD application suppresses the down-growth of junctional epithelium onto dental root surfaces, a process that frequently interferes with the formation of new connective tissue attachments.

L18 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2001 ACS
1999:795994 Document No. 132:31744 Gene probes used for genetic profiling in
healthcare screening and planning. Roberts, Gareth Wyn (Genostic Pharma Ltd., UK). PCT Int. Appl. WO 9964627 A2 19991216, 745 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-GB1780 19990604. PRIORITY: GB 1998-12099 19980606; GB 1998-13291 19980620; GB 1998-13611 19980624; GB 1998-13835 19980627; GB
1998-14110 19980701; GB 1998-14580 19980707; GB 1998-15438 19980716; GB 1998-15576 19980718; GB 1998-15574 19980718; GB 1998-16085 19980724; GB 1998-16086 19980724; GB 1998-16921 19980805; GB 1998-17097 19980807; GB 1998-17200 19980808; GB 1998-17632 19980814; GB 1998-17943 19980819.

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating
that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response.
In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol.
states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified
in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies
which comprises of the identification of the core group of genes and their
sequence variants required to provide a broad base of clin. prognostic information - "genostics". The "Genostic.RTM." profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most
in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of

persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

L18 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2001 ACS
1999:795993 Document No. 132:31743 Gene probes used for genetic profiling in
healthcare screening and planning. Roberts, Gareth Wyn (Genostic Pharma Limited, UK). PCT Int. Appl. WO 9964626 A2 19991216, 149 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-GB1779 19990604. PRIORITY: GB 1998-12098 19980606; GB 1998-28289 19981223.

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating
that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response.
In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol.
states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified
in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies.

L18 ANSWER 8 OF 11 MEDLINE DUPLICATE 3
1998046690 Document Number: 98046690. Detection of **apoptosis**-related factors and apoptotic cells in ameloblastomas: analysis by immunohistochemistry and an in situ DNA nick end-labelling method. Kumamoto H. (Department of Oral Pathology, Tohoku University School of Dentistry, Sendai, Japan.) JOURNAL OF ORAL PATHOLOGY AND MEDICINE, (1997 Oct) 26 (9) 419-25. Journal code: JRF. ISSN: 0904-2512. Pub. country: Denmark. Language: English.

AB To clarify the possible role of **apoptosis** in odontogenic epithelium, **apoptosis**-related factors and apoptotic cells were examined by immunohistochemistry and an in situ DNA nick end-labelling method. Expression of bcl-2 protein was detected in both normal and neoplastic odontogenic epithelium, whereas expression of p53 protein was detected only in neoplastic but not in normal odontogenic epithelium. The

prevalence of cases positive for Lewis(y) antigen in ameloblastomas was significantly lower than in **enamel** organs. Correlation between these factors and apoptotic cells presented by an in situ DNA nick end-labelling method was not clear. The number of apoptotic cells in ameloblastomas was significantly greater than in normal odontogenic epithelium, and apoptotic reactions in the granular cell type ameloblastoma tended to be more frequently detected than in other types of ameloblastomas. These results suggested that apoptotic **cell death** might play an important role in oncogenesis and/or tissue differentiation in odontogenic epithelium.

L18 ANSWER 9 OF 11 MEDLINE

95308550 Document Number: 95308550. Detection of the **apoptosis**-suppressing oncoprotein bcl-2 in ameloblastomas. Gao Y; Yang L; Zhu X. (Department of Oral Pathology, Stomatological College, Fourth Military Medical University, Xi'an.) CHUNG-HUA PING LI HSUEH TSA CHIH [CHINESE JOURNAL OF PATHOLOGY], (1995 Apr) 24 (2) 78-9. Journal code: CZQ. ISSN: 0529-5807. Pub. country: China. Language: Chinese.

AB The product of **apoptosis**-suppressing oncoprotein bcl-2 can block **apoptosis** and result in the development of **tumors**. In this study, the expression of bcl-2 was observed in 40 cases of ameloblastomas by immunohistochemical staining. The results showed that the epithelium and reduced **enamel** epithelium of the **enamel** organ, the odontoblasts, basal cells of odontogenic cysts, normal oral epithelium and 90% (36/40) of ameloblastomas were positive for bcl-2, indicating that the expression of bcl-2 in odontogenic epithelium may be related to the degree of differentiation and proliferation of cells, the overexpression of bcl-2 may be associated with the development of ameloblastoma.

L18 ANSWER 10 OF 11 MEDLINE DUPLICATE 4

95031718 Document Number: 95031718. Immunohistochemical demonstration of bcl-2 protein in human tooth germs. Slootweg P J; de Weger R A. (Department of Pathology, University Hospital, Utrecht, The Netherlands.)

ARCHIVES OF ORAL BIOLOGY, (1994 Jul) 39 (7) 545-50. Journal code: 83M. ISSN: 0003-9969. Pub. country: ENGLAND: United Kingdom. Language: English.

AB This study sought to detect patterns of bcl-2 protein expression that could provide more insight into the cellular dynamics of tooth development. As bcl-2 serves to prevent **cell death**, its occurrence in odontogenic tissues might be helpful in identifying cell populations from which odontogenic **tumours** may arise. The bcl-2 protein was found only in the epithelial part of the tooth germ and was present in all parts of the **enamel** organ except the ameloblast. This suggests that bcl-2 protein plays a part in maintaining the viability of the **enamel** organ. The presence of bcl-2 in the fully matured tooth germ and adjacent dental lamina might indicate that epithelial odontogenic **tumours** may originate from various parts of the **enamel** organ.

L18 ANSWER 11 OF 11 MEDLINE DUPLICATE 5
76020545 Document Number: 76020545. Ultrastructural study of amyloid material in the calcifying epithelial odontogenic **tumor**. Page D L; Weiss S W; Eggleston J C. CANCER, (1975 Oct) 36 (4) 1426-35. Journal code: CLZ. ISSN: 0008-543X. Pub. country: United States. Language: English.

AB A typical calcifying epithelial odontogenic **tumor** of the maxilla was examined with the electron microscope. The **tumor** cell resembles the ameloblast at an early stage of **enamel** deposition. Formation of extracellular amyloid masses probably proceeds both by active cellular secretion and **cell death**, each process adding similar granulofibrillar material to these masses, which tend to calcify. The amyloid masses are probably a relatively homogenous protein material and represent a specific **tumor** cell product. Further characterization of this **neoplasm** must include chemical and physical studies of this extracellular **tumor** product, which is an amyloid material because of classic staining characteristics.

'IN' IS NOT A VALID FIELD CODE
L19 474 FILE MEDLINE
L20 136 FILE CAPLUS
L21 490 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L22 378 FILE EMBASE
L23 8 FILE WPIDS
'IN' IS NOT A VALID FIELD CODE
L24 589 FILE SCISEARCH
'IN' IS NOT A VALID FIELD CODE
L25 0 FILE NTIS

TOTAL FOR ALL FILES
L26 2075 HAMMARSTROM L?/AU,IN

'IN' IS NOT A VALID FIELD CODE
L27 21 FILE MEDLINE
L28 9 FILE CAPLUS
L29 15 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L30 10 FILE EMBASE
L31 2 FILE WPIDS
'IN' IS NOT A VALID FIELD CODE
L32 20 FILE SCISEARCH
'IN' IS NOT A VALID FIELD CODE
L33 0 FILE NTIS

TOTAL FOR ALL FILES
L34 77 LYNGSTADAAS S?/AU,IN

'IN' IS NOT A VALID FIELD CODE
L35 23 FILE MEDLINE

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L36          26 FILE CAPLUS
L37          28 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L38          16 FILE EMBASE
L39           6 FILE WPIDS
'IN' IS NOT A VALID FIELD CODE
L40          28 FILE SCISEARCH
'IN' IS NOT A VALID FIELD CODE
L41           0 FILE NTIS

TOTAL FOR ALL FILES
L42         127 GESTRELIUS S?/AU,IN

=> s l42 and l34 and l26
L43           1 FILE MEDLINE
L44           1 FILE CAPLUS
L45           0 FILE BIOSIS
L46           0 FILE EMBASE
L47           0 FILE WPIDS
L48           0 FILE SCISEARCH
L49           0 FILE NTIS

TOTAL FOR ALL FILES
L50           2 L42 AND L34 AND L26

=> s l50 not l17
L51           1 FILE MEDLINE
L52           0 FILE CAPLUS
L53           0 FILE BIOSIS
L54           0 FILE EMBASE
L55           0 FILE WPIDS
L56           0 FILE SCISEARCH
L57           0 FILE NTIS

TOTAL FOR ALL FILES
L58           1 L50 NOT L17

=> d cbib abs

L58  ANSWER 1 OF 1  MEDLINE
2001150350  Document Number: 21087066.  Emdogain--periodontal regeneration
     based on biomimicry. Gestrelius S; Lyngstadaas S P;
     Hammarstrom L. (Biora AB, MEDEON, Malmo SE 205 12, Sweden..
     stina.gestrelius@biora.se) . CLINICAL ORAL INVESTIGATIONS, (2000 Jun) 4
     (2) 120-5.  Ref: 42. Journal code: C3I. ISSN: 1432-6981. Pub. country:
     Germany: Germany, Federal Republic of. Language: English.
AB   Biomimicry has been introduced as a term for innovations inspired by
     nature [1]. Such innovations may appear in almost every part of modern
     society. This review on the effects of enamel matrix proteins on the
     formation of cementum and the development of emdogain for regeneration of
     periodontal tissues lost due to periodontitis shows an example of
     biomimicry in dentistry. Findings from clinical and laboratory
     investigations are summarized and the biological basis for enamel
     matrix-induced periodontal regeneration is discussed.
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=> s (dent? or periodont?)
L59      303500 FILE MEDLINE
L60       50919 FILE CAPLUS
L61      137342 FILE BIOSIS
L62       47329 FILE EMBASE
L63       36133 FILE WPIDS
L64       87262 FILE SCISEARCH
L65        6607 FILE NTIS

TOTAL FOR ALL FILES
L66      669092 (DENT? OR PERIODONT?)

=> s l66 and (apoptos? or capase or cell death or clonal delet?)
L67         556 FILE MEDLINE
L68         421 FILE CAPLUS
L69        1352 FILE BIOSIS
L70         482 FILE EMBASE
L71          25 FILE WPIDS
L72         630 FILE SCISEARCH
L73           0 FILE NTIS

TOTAL FOR ALL FILES
L74        3466 L66 AND (APOPTOS? OR CAPASE OR CELL DEATH OR CLONAL DELET?)

=> s l74 and (neoplasm? or cancer or tumour or tumor)
L75          32 FILE MEDLINE
L76          24 FILE CAPLUS
L77         281 FILE BIOSIS
L78          20 FILE EMBASE
L79          12 FILE WPIDS
L80          35 FILE SCISEARCH
L81           0 FILE NTIS

TOTAL FOR ALL FILES
L82         404 L74 AND (NEOPLASM? OR CANCER OR TUMOUR OR TUMOR)

=> s l82 and (hammarstrom l? or lyngstadaas s? or gestrelius s?)/au,in
'IN' IS NOT A VALID FIELD CODE
L83           0 FILE MEDLINE
L84           0 FILE CAPLUS
L85           0 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L86           0 FILE EMBASE
L87           0 FILE WPIDS
'IN' IS NOT A VALID FIELD CODE
L88           0 FILE SCISEARCH
'IN' IS NOT A VALID FIELD CODE
L89           0 FILE NTIS

TOTAL FOR ALL FILES
L90           0 L82 AND (HAMMARSTROM L? OR LYNGSTADAAS S? OR GESTRELIUS
S?)/AU,I
               N
```